Impact of childhood obesity treatment on body composition and metabolic profile

Marja Kalavainen, Pauliina Utriainen, Esko Vanninen, Matti Korppi, Outi Nuutinen
Kuopio, Finland

Background: **Childhood obesity is associated with adverse changes in cardiometabolic risk factors. A family-oriented group program stressing a health-promoting lifestyle has been more effective than routine counselling in the treatment of obesity in school children. The aim of the present study was to compare the impact of group program and routine councelling on body composition and metabolic profile, and to evaluate the associations of changes in adiposity with levels of cardiometabolic risk factors.**

Methods: **Seventy obese prepubertal children were randomized into family-oriented group program (15 sessions for parents and children) and routine counselling (2 appointments for children). Body mass index (BMI), body composition and different metabolic risk factors were assessed before and after the 6-month intervention.**

Results: **Waist/height decreased more in the children attending the group treatment, but there were no significant differences between treatment arms in the changes of metabolic risk factors. When the arms were analyzed as combined, serum triglycerides decreased significantly if BMI standard deviation score (BMI-SDS) decreased ≥0.5. Serum fasting insulin decreased if BMI-SDS decreased ≥0.25.**

Conclusions: **Obesity-related metabolic risk factors reduced in prepubertal children if BMI-SDS decreased substantially. This result was not dependent on which intervention, family-oriented group program or routine counselling, was used.**

World J Pediatr 2012;8(1):31-37

Key words: **body composition; childhood obesity; metabolism**

Author Affiliations: Departments of Pediatrics and Clinical Nutrition, Kuopio University Hospital, Kuopio, Finland (Kalavainen M); Department of Pediatrics, Kuopio University Hospital and University of Eastern Finland, Finland (Utriainen P); Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital and University of Eastern Finland, Finland (Vanninen E); Pediatric Research Center, Tampere University and University Hospital, Tampere, Finland (Korppi M); Institute of Public Health and Clinical Nutrition, Department of Clinical Nutrition, University of Eastern Finland, Kuopio, Finland (Nuutinen O)

Corresponding Author: Matti Korppi, Pediatric Research Center, Tampere University and University Hospital, FinMed-3 building, FIN-33014 Tampere University, Finland (Tel: +358-3-35518407; Fax: +358-3-35538420; Email: matti.korppi@uta.fi)

doi: 10.1007/s12519-011-0324-2

Introduction

Childhood obesity is associated with adverse changes in cardiometabolic risk factors.[1] In prepubertal obese children, for example, serum triglycerides (TG) are increased, high density lipoprotein cholesterol (HDL-C) is decreased, and insulin sensitivity is impaired.[2] Waist circumference, in particular, is a predictor of metabolic abnormalities.[3] Childhood obesity at age of 7-13 years was found to be associated with coronary heart disease in adulthood in a large population-based study.[4]

Randomized controlled trials on treatment of childhood obesity have documented conflicting impacts on cardiometabolic risk factors.[5-8] In a German non-randomized study, metabolic abnormalities improved if the decrease in body mass index standard deviation score (BMI-SDS) was >0.5.[9,10]

We have previously reported that a family-oriented group program stressing a health-promoting lifestyle was more effective[11] and also more costly[12] than was routine counselling in the treatment of 7-9 year old obese children. The aim of the present study was to compare the effects of these two programs on children's body composition and metabolic profile. In addition, the associations between changes in children's adiposity and levels of cardiometabolic risk factors were evaluated.

Methods

Subjects and interventions

The detailed study design has been described previously.[11] Briefly, families with 7-9 year old obese

children (weight for height >120%, calculated from parent-reported weights and heights) were recruited, and 70 children (28 boys, 42 girls) participated in the study. At baseline, the mean age of the children was 8.1 years (SD 0.8; range 6.6-9.7), the mean weight for height 142% (14.4; 115-182), the mean BMI 23.2 (2.5; 18.7-30.9) and the mean BMI-SDS 2.6 (SD 0.6; 1.3-3.8). The children were randomized into two 6-month programs: routine program (n=35) and group program (n=35). The routine program was modified from the current counselling practice for obese children in school health care, and consisted of two standardized individual appointments.

The group program consisted of 14 sessions held separately to parents and children, and one session held together.[11] There were 5 groups, each consisting of 7 children and their parents. The duration of each session was 90 minutes, and the first 10 were held weekly and the next 5 every two weeks. The participation rate for the sessions was 87% in both the parents and children. Each session had an own, physical activity and healthy lifestyle promoting theme. The children's session consisted of interactive functional activities (e.g., games, tasting of vegetables and fruits, preparing of foods and drinks), and parent's sessions consisted of lectures and discussions. In addition, the programs contained homework; parents were provided with treatment manuals and children with workbooks. Most lifestyle changes were intended for the entire family.

Informed consent was obtained from the parents. The study was performed according to the principles of the *Declaration of Helsinki*, and approved by the Ethics Committee of Kuopio University and Kuopio University Hospital.[11]

As published previously, the group program was more costly[12] and more effective[11] when assessed by changes in weight for height (on average, 6.8% *vs.* 1.8% reduction, P=0.001), BMI (on average, 0.8 *vs.* 0.0 reduction, P=0.003) and BMI-SDS (on average, 0.3 *vs.* 0.2 reduction, P=0.022) between the baseline and the end of the intervention.

Measurements

The children were examined before and after the 6-month intervention. All measurements were performed in the morning after 11-13 hours fasting using a standard protocol.

Height and waist circumference were measured 3 times, weight was measured 2 times, and the average values were used for analyses. Height was measured to the nearest 0.1 cm using a Harpenden Stadiometer (Practical Metrology, UK) and weight (light underwear included) to the nearest 0.1 kg using an electronic scale (Seca Vogel & Halke, Germany).

The change in the weight for height, that is a percentage deviation of weight from the median height-related gender-specific weight, was assessed by the Pediator program.[13] Weight for height is in routine use through the whole health care system in our country, and population-based gender-specific growth charts from birth to the age of 18 years are available.[14,15] BMI was calculated using the formula: weight (kg) / [height (m)]^2. BMI-SDS was computed by an automatic calculator.[16] The calculator uses the British gender-specific growth reference from 1990, produced by the LMS method,[17,18] and revised in 1996.[19] The LMS method summarizes the distribution of BMI at each age by its median (M), coefficient of variation (S), and a measure of skewness expressed as a Box-Cox power (L) required to transform the data to normality.[20] BMI-SDS changes were categorized into 4 groups: any BMI-SDS increase, BMI-SDS decrease <0.25, BMI-SDS decrease ≥0.25 but <0.5, and BMI-SDS decrease ≥0.5.[9]

Waist circumference was measured at the midpoint between the lateral iliac crest and the lowest rib to the nearest 0.5 cm using a flexible tape. Waist/height ratio was calculated by dividing waist circumference (cm) by height (cm).[21]

Fat mass and lean body were assessed by bioelectrical impedance analysis with Inbody 3.0® (Biospace, Seoul, South Korea)[22] for subjects in upright position after voiding.

Tanner pubertal status was assessed.[23] The diameters of areola and palpable breast tissue in girls and testicular length in boys were measured with a ruler. Children with Tanner stage G1/M1 were recorded as prepubertal and with G2-5/M2-5 as pubertal.[23]

Total plasma cholesterol (TC), HDL-C, low-density lipoprotein cholesterol (LDL-C) and TG concentrations were determined by enzymatic methods (Thermo Electron Co., Vantaa, Finland) and plasma glucose concentration was determined by a hexokinase method (Thermo Electron Co., Vantaa, Finland). Serum insulin was analyzed with a time-resolved immunofluorometric method by AutoDelfia (PerkinElmer Life and Analytical Sciences Wallac Oy, Turku, Finland). Insulin resistance (HOMA-IR) was calculated using the following formula: [insulin (mU/L) × glucose (mmol/L)]/22.5.[24]

Blood pressure was measured automatically on the right arm of a seated child by a Dinamap™ XL monitor with appropriate-sized cuffs. Three measurements were performed after rest for five minutes, and the results of the second and third were averaged for analysis.

Missing data

The baseline values were used also as the post-treatment values for one child in the group program

and for one child in the routine program. In addition, one child refused blood tests and one child refused blood pressure measurements (group program). One additional child (routine program), who drank juice (containing 10 g carbohydrates) for dizziness, was excluded from glucose metabolism analyses and one child was excluded from TG analyses because of marked hypertriglyceridemia (6.18 mmol/L) (group program).

Control children

The control group comprised healthy prepubertal children with normal weight (mean weight for height 100%, SD 7.9), matched for age (mean 7.8 years, SD 0.7) and gender (49 girls, 13 boys) with the study children. The children of the control group were originally collected for a study on premature adrenarche (control group of the study) from the same area.[25] Their bioimpedance, laboratory and blood pressure measurements were used as reference values. The methods were similar in the study and control groups, except for plasma glucose, which was measured by a glucose oxidase method (Clarke Electrode, Rapidlab 865/1265; Bayer,Tarrytown, NY). The glucose hexokinase and glucose oxidase methods were calibrated to match with each other. In addition, blood pressure was analyzed with a standard sphygmomanometer in the control group. Waist and waist/height data in 87 German 8-year-old girls were used as reference values.[26]

Statistical analysis

Data were analyzed using the SPSS 16.0 software (SPSS Inc., Chicago, IL). Continuous variables were tested for normality with the Saphiro-Wilk test, and logarithmic (log) transformation was performed if necessary. Baseline differences of continuous variables and differences in changes between the treatment arms were analyzed by the independent samples *t* test or the Mann-Whitney *U* test. Differences within BMI-SDS change categories were analyzed by the paired-samples *t* test or Wilcoxon's signed-rank test. Fisher's exact test was used for analysis of discrete variables.

Results

Sixty-three children were prepubertal before intervention, and 7 were at an early puberty. Nine children entered puberty during the intervention. There were no significant differences between the study groups in pre- or post-intervention pubertal status (Data not shown).

At baseline, the study children had in mean a higher fat mass (13.7 *vs.* 4.1 kg, $P<0.001$) and lean body mass (28.1 *vs.* 22.0 kg, $P<0.001$) compared with the normal-weight control children (Table 1). In the laboratory values, the study children had lower HDL-C (1.15 *vs.* 1.50 mmol/L, $P<0.001$), and higher LDL-C (2.73 *vs.* 2.47 mmol/L, $P=0.012$), TG (0.88 *vs.* 0.58 mmol/L, $P<0.001$), glucose (5.2 *vs.* 4.8 mmol/L, $P<0.001$), fasting insulin (9.4 *vs.* 4.1 mU/L, $P<0.001$) and HOMA-IR (2.17 *vs.* 1.07, $P<0.001$) compared with the control children (Tables 1 and 2).

At baseline, there were no significant differences in the body composition or in the metabolic profile between the children in the two obesity treatment arms. During the intervention, the decrease in waist/height was greater in the group program (mean 0.02, 95% CI 0.01-0.03) than in the routine program (0.01, 0.00-0.02), respectively (Table 1). However, there were no significant differences in the changes of metabolic indicators between the two programs (Tables 1 and 2). Likewise, the changes were not related to BMI-SDS at baseline (Data not shown).

Within the intervention arms, the children in the group program showed significant decreases in fat

Table 1. Efficacy of the 6-month routine and group programs, expressed as changes in body composition and blood pressure

Outcome measures	Normal weight children†	Study children in the routine program (*n*=35)			Study children in the group program (*n*=35)			*P* value between programs
	M (SD)	Baseline M (SD)	After M (SD)	Change M [95%CI]	Baseline M (SD)	After M (SD)	Change M [95%CI]	
Fat mass (kg)	4.1 (1.5)	13.1 (3.9)	12.9 (4.5)	–0.2 [–0.8 to 0.5]	14.4 (4.3)	13.5 (4.2)	–0.9 [–1.5 to –0.2]	0.147§
Lean body mass (kg)	22.0 (2.6)	27.4 (3.7)	29.3 (4.2)	+1.9 [+1.4 to 2.4]	28.8 (5.2)	30.1 (5.3)	+1.4 [+1.0 to 1.8]	0.096‖
Waist (cm)	58.7 (6.5)	75.3 (6.1)	76.1 (6.9)	+0.8 [–0.4 to 2.0]	77.3 (7.4)	76.7 (7.4)	–0.7 [–1.7 to 0.4]	0.062§
Waist/height	0.44 (0.04)	0.57 (0.04)	0.56 (0.04)	–0.01 [–0.02 to 0.00]	0.57 (0.04)	0.55 (0.04)	–0.02 [–0.03 to –0.01]	0.047§
SBP (mmHg)*	98.8 (7.5)	108.1 (6.8)	108.1 (7.1)	0.0 [–1.7 to 1.6]	110.8 (8.3)‡	109.9 (9.3)‡	–0.9 [–3.2 to 1.3]‡	0.503¶
DBP (mmHg)*	59.3 (7.2)	55.2 (6.5)	54.5 (6.6)	–0.7 [–2.9 to 1.4]	55.2 (6.5)‡	55.3 (6.4)‡	+0.2 [–1.3 to 1.6]‡	0.489§

M: mean; SD: standard deviation; CI: confidence interval; SBP: systolic blood pressure; DBP: diastolic blood pressure. *: For normal weight children standard sphygmomanometer, in the present study automated device; †: Finnish normal-weight children, matched for age and gender, *n*=62 (see control children in methods); for waist and waist/height German 8-year-old girls [25], *n*=87; ‡: *n*=34; §: Independent samples *t* test; ‖: the Mann-Whitney *U* test; ¶: Independent samples *t* test with the logarithmic transformation.

Table 2. Efficacy of the 6-month routine and group programs, expressed as changes in laboratory values

Outcome measures	Normal-weight children*	Study children in the routine program (n=35)			Study children in the group program (n=35)			P value between programs
		Baseline M (SD)	After M (SD)	Change M [95%CI]	Baseline M (SD)	After M (SD)	Change M [95%CI]	
TC (mmo/L)	4.2 (0.6)	4.2 (0.7)	4.4 (0.8)	+0.1 [–0.1 to 0.3]	4.3 (0.6)†	4.5 (0.7)†	+0.2 [0.0 to 0.4]†	0.493§
LDL-C (mmol/L)	2.47 (0.52)	2.77 (0.69)	2.77 (0.75)	+0.01 [–0.17 to 0.18]	2.69 (0.55)†	2.72 (0.56)†	+0.03 [–0.09 to 0.14]†	0.500§
HDL-C (mmol/L)	1.50 (0.31)	1.12 (0.27)	1.20 (0.34)	+0.07 [0.00 to 0.15]	1.18 (0.27)†	1.30 (0.24)†	+0.12 [+0.07 to 0.18]†	0.317‖
TG (mmol/L)	0.58 (0.20)	0.85 (0.40)	0.83 (0.49)	–0.02 [–0.15 to 0.10]	0.91 (0.56)‡	0.68 (0.24)‡	–0.23 [–0.39 to –0.07]‡	0.093¶
F-glucose (mmol/L)	4.8 (0.3)	5.2 (0.3)†	5.3 (0.3)†	+0.1 [0.0 to 0.2]†	5.2 (0.3)†	5.2 (0.4)†	0.0 [–0.1 to 0.1]†	0.145‖
F-insulin (mU/L)	4.1 (1.9)	8.5 (4.4)†	8.6 (4.1)†	0.0 [–1.5 to 1.6]†	10.2 (5.8)†	8.6 (5.3)†	–1.6 [–3.1 to –0.1]†	0.142¶
HOMA-IR	1.07 (0.38)	1.96 (1.03)†	2.03 (1.01)†	+0.07 [–0.32 to 0.47]†	2.39 (1.43)†	2.01 (1.27)†	–0.37 [–0.74 to –0.01]†	0.113¶

M: mean; SD: standard deviation; CI: confidence interval; TC: total cholesterol; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; TG: triglycerides; F: fasting; HOMA-IR: homeostasis model assessment for insulin resistance. *: Finnish normal-weight children, matched for age and gender, n=62 (see control children in methods); †: n=34; ‡: n=33; §: Mann-Whitney U test; ‖: Independent samples t test; ¶: Independent samples t test with the logarithmic transformation.

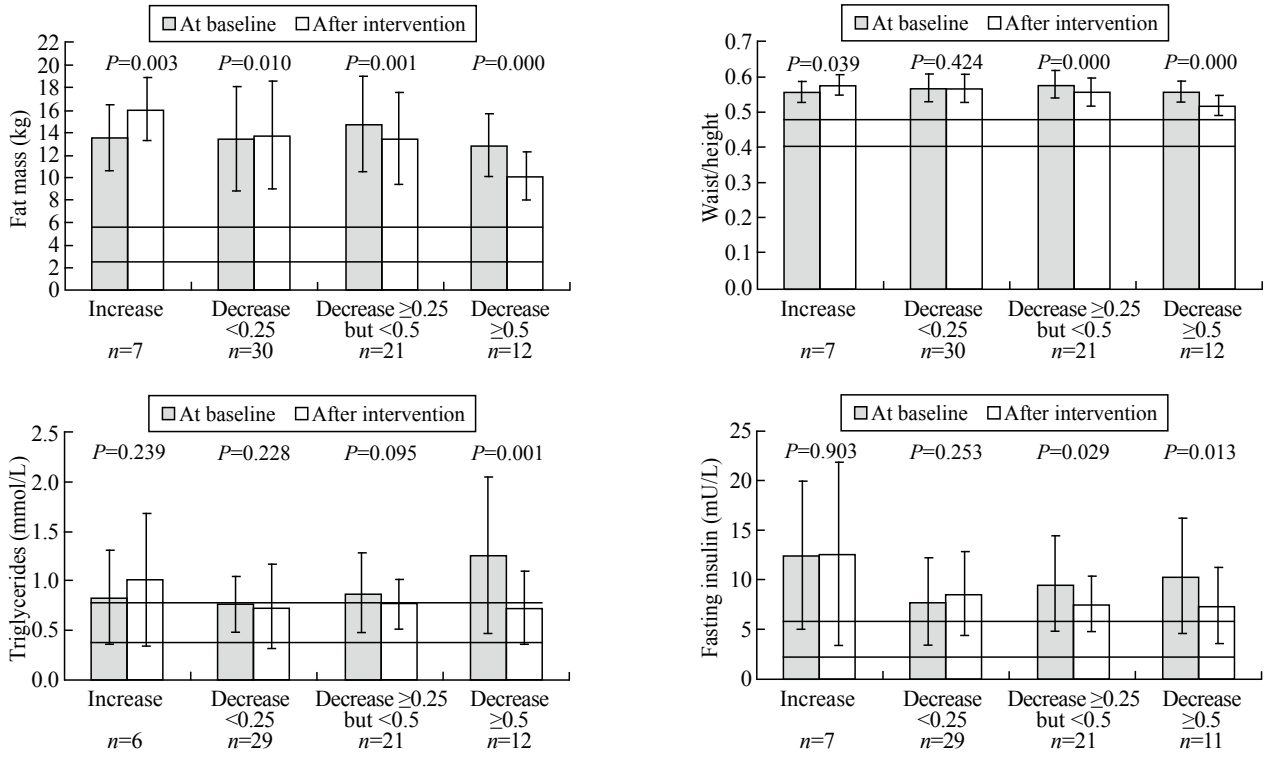

Fig. Associations between selected measures of body composition and metabolic indicators (means ± 1 SD) according to categories of the change in BMI-SDS during the 6-month treatment. The normal values (means ± 1 SD) based on normal-weight controls are marked with horizontal lines. Independent samples t test and the Mann-Whitney U test were used in statistical analyses.

mass (mean 0.9 kg, 95% CI 0.2-1.5), waist/height (0.02, 0.01-0.03), TG (0.23 mmol/L, 0.07-0.39), fasting insulin (1.6 mU/L, 0.1-3.1) and HOMA-IR (0.37, 0.01-0.74), and increases in lean body mass (1.4 kg, 1.0-1.8), TC (0.2 mmol/L, 0.0 to 0.4) and HDL-C (0.12 mmol/L, 0.07-0.18) (Tables 1 and 2). In contrast, children in the routine program showed an increase in lean body mass (1.9 kg, 1.4-2.4) and an unexpected increase in fasting glucose (0.1 mmol/L, 0.0-0.2).

The changes in body composition and metabolic profile were analyzed jointly for all children in the four BMI-SDS change categories (BMI-SDS increase, a BMI-SDS decrease <0.25, a BMI-SDS decrease ≥0.25 but <0.5 and a BMI-SDS decrease ≥0.5). The changes in body composition and metabolic indicators with a major deviation from the control children are presented in the Fig. Declines in fat mass, waist/height and fasting insulin were seen in the children with a BMI-SDS decrease of ≥0.25. A decline in TG was seen only in the children with a BMI-SDS decrease of ≥0.5. The associations between HDL-cholesterol and BMI-SDS changes were small and inconsistent (Data not shown).

Discussion

There were two main results in the present study. First, the children in the group treatment program lost more abdominal adiposity, as estimated by the waist/height, than those in the routine program, but there were no differences in metabolic indicators between the programs. As earlier published, the group program was more costly[12] and more effective[11] than was the routine program in the treatment of obesity, when the effectiveness was assessed by weight for height and BMI-SDS decreases. Second, decreases in BMI-SDS were associated with beneficial changes in metabolic indicators when both treatment arms were combined.

Before intervention, all laboratory measures of the obese study children were, on average, within normal limits. When metabolic indicators of the study children were compared with the normal-weight control children, HDL-C levels were substantially lower, and TG, fasting insulin and HOMA-IR were higher. Thus, our findings are in accordance with a Spanish study,[2] in which prepubertal obese children had elevated TG and fasting insulin, and diminished HDL-C compared with age-matched normal-weight children. In the present study, the blood pressure values differed marginally from the control children, but were in line with the values in the children of same age from Great Britain.[27]

In the present study, the children in the group program lost more abdominal adiposity, as estimated by the waist/height, than those in the routine program, but there were no other significant differences in metabolic indicators. The impact of obesity treatment on metabolic indices in prepubertal children was evaluated in an Australian 6-month randomized controlled study in 111 children aged on average 8.2 years.[7] At 12 months, BMI-SDS was decreased in all three treatment arms (10% in parenting-skills training + intensive lifestyle education, 5% in parenting-skills training alone, and 5% in waiting list controls), but there were no differences in metabolic indices between the treatment arms or between baseline and 12 months.[7]

In contrast to the present and the Australian study,[7] some obesity treatment trials have shown beneficial changes in children's TC or LDL-C concentrations,[5,6,8] blood pressure,[5] fasting insulin[8] and insulin resistance.[8] Because of the different age groups, designs and outcome measures, comparisons of the results must be done cautiously. For example, children have been older and changes in BMI-SDS have been larger[5,6,8] than in the present and the Australian study.[7]

In the present study, weight loss was associated with beneficial changes in TGs when BMI-SDS decreased >0.5, and in fasting insulin when BMI-SDS decreased >0.25. There are conflicting results on how large BMI-SDS decreases are associated with beneficial metabolic impacts. In a German non-randomized 1-year obesity treatment study in 130 children aged 11 years on average with mean BMI-SDS 2.5, an increase in HDL-C and decreases in TG, LDL-cholesterol, HOMA-IR and blood pressure were seen when the BMI-SDS decrease was >0.5.[9] On the other hand, a recent 1-year prospective cohort study in 88 adolescents (median age 12.4 years, mean BMI-SDS 3.23) suggested improvements in metabolic indices with a BMI-SDS decrease of 0.25.[28] In that study, a BMI-SDS decrease of 0.25 improved TG, TC/HDL-ratio, HOMA-IR and blood pressure, and a BMI-SDS decrease of 0.50 was associated with greater metabolic benefits.[28]

The clinical significance of the reduced adiposity and associated improvement in metabolic profile in children is uncertain. In adults with impaired glucose tolerance, type 2 diabetes can be prevented or at least postponed by modest weight loss combined with lifestyle changes.[29] In children, there is an association between obesity, cardiometabolic risk factors and increased carotid intima-media thickness,[30,31] which seemed to be reversible if marked weight loss (BMI-SDS decrease >0.5) occurred.[30]

There are three strengths in this prospective, controlled, and randomized study. First, our study population consisted of mainly prepubertal children, and so, the presence or absence of puberty did not confound the results. Recording pubertal stage is important since children undergo major metabolic changes during puberty.[32] Second, we had a population-based control group from the same area consisting of normal-weight children matched for age and gender.[25] And third, all measurements were performed using standard protocols, and children's adiposity was assessed with bioelectrical impedance, which is a well-established method for body composition assessment.[33]

The study was evidently under-powered to detect small differences in the metabolic indicators between the subgroups. For example, only 12 children reached a BMI-SDS decrease ≥0.5. Moreover, we have no follow-up data on body composition and metabolic indicators beyond the end of intervention.

In the clinical follow-up, the reduction in weight for height was not significant but the reduction in BMI-SDS was significant though slight 2 years (mean reduction 0.2 SD) and 3 years (mean reduction 0.3 SD) after intervention.[34] There were no differences between children attending the group treatment and routine counselling. Thus, long-term programs are needed for long-term benefits, such as repeated group sessions at long intervals after intensive interventions or other extended regular contacts, for instance, using a telemedicine support program.[35]

In conclusion, the children in the group program lost more abdominal adiposity than those in the routine program, but there were no differences in metabolic indicators between the treatment arms. Although there was no clear threshold for favorable BMI-SDS decrease, serum triglycerides decreased significantly if BMI-SDS decreased ≥0.5, and serum fasting insulin decreased if BMI-SDS decreased ≥0.25.

Funding: This research was supported in part by grants from Kuopio University Hospital, the Scientific Foundation of Finnish Association of Academic Agronomists, Finnish Cultural Foundation of Northern Savo, Juho Vainio Foundation, Ministry of Social Affairs and Health, Social Insurance Institution, and the Finnish Cultural Foundation.

Ethical approval: The Ethics Committee of Kuopio University and University Hospital accepted the study. An informed consent was obtained from the parents of the children.

Competing interest: None of the authors has any competing interests.

Contributors: Kalavainen M collected the data, made the analyses and was primarily responsible for the writing of the manuscript. Utriainen P collected and analyzed the controls and participated in the analyses and writing. Vanninen E was responsible for the methods of clinical physiology and interpreted the data and participated in the writing. Korppi M participated in the planning of the study, interpreted the statistics and participated in the writing. Nuutinen O was responsible for the planning and designing of the study and participated in the interpretation of the results and writing of the manuscript.

References

1 Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L, et al. Health consequences of obesity. Arch Dis Child 2003;88:748-752.
2 Garcés C, Gutierrez-Guisado J, Benavente M, Cano B, Viturro E, Ortega H, et al. Obesity in Spanish schoolchildren: relationship with lipid profile and insulin resistance. Obes Res 2005;13:959-963.
3 Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The metabolic syndrome in children and adolescents. Lancet 2007;369:2059-2061.
4 Baker JL, Olsen LW, Sorensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med 2007;357:2329-2337.
5 Jiang JX, Xia XL, Greiner T, Lian GL, Rosenqvist U. A two year family based behaviour treatment for obese children. Arch Dis Child 2005;90:1235-1238.
6 Nemet D, Barkan S, Epstein Y, Friedland O, Kowen G, Eliakim A. Short- and long-term beneficial effects of a combined dietary-behavioral-physical activity intervention for the treatment of childhood obesity. Pediatrics 2005;115:e443-e449.
7 Golley RK, Magarey AM, Baur LA, Steinbeck KS, Daniels LA. Twelve-month effectiveness of a parent-led, family-focused weight-management program for prepubertal children: a randomized, controlled trial. Pediatrics 2007;119:517-525.
8 Savoye M, Shaw M, Dziura J, Tamborlane WV, Rose P, Guandalini C, et al. Effects of a weight management program on body composition and metabolic parameters in overweight children: a randomized controlled trial. JAMA 2007;297:2697-2704.
9 Reinehr T, Andler W. Changes in the atherogenic risk factor profile according to degree of weight loss. Arch Dis Child 2004;89:419-422.
10 Reinehr T, Kiess W, Kapellen T, Andler W. Insulin sensitivity among obese children and adolescents, according to degree of weight loss. Pediatrics 2004;114:1569-1573.
11 Kalavainen MP, Korppi MO, Nuutinen OM. Clinical efficacy of group-based treatment for childhood obesity compared with routinely given individual counselling. Int J Obes (Lond) 2007;31:1500-1508.
12 Kalavainen M, Karjalainen S, Martikainen J, Korppi M, Linnosmaa I, Nuutinen O. Cost-effectiveness of routine and group programs for treatment of obese children. Pediatr Int 2009;51:606-611.
13 Tilator Oy Ltd. Pediator software for growth data [computer program]. Version Release 6.7.4. Säkylä, Finland, 2004.
14 Pere A. Comparison of two methods for transforming height and weight to normality. Ann Hum Biol 2000;27:35-45.
15 Sorva R, Lankinen S, Tolppanen EM, Perheentupa J. Variation of growth in height and weight of children. II. After infancy. Acta Paediatr Scand 1990;79:498-506.
16 Northwest Institute for Bio-Health Informatics: child obesity calculator using British 1990 growth reference data. http://www.phsim.man.ac.uk/ (accessed January 18, 2007).
17 Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. Arch Dis Child 1995;73:25-29.
18 Freeman JV, Cole TJ, Chinn S, Jones PR, White EM, Preece MA. Cross-sectional stature and weight reference curves for the UK, 1990. Arch Dis Child 1995;73:17-24.
19 Cole TJ, Freeman JV, Preece MA. British 1990 growth reference centiles for weight, height, body mass index and head circumference fitted by maximum penalized likelihood. Stat Med 1998;17:407-429.
20 Cole TJ. The LMS method for constructing normalized growth standards. Eur J Clin Nutr 1990;44:45-60.
21 McCarthy HD, Ashwell M. A study of central fatness using waist-to-height ratios in UK children and adolescents over two decades supports the simple message—'keep your waist circumference to less than half your height'. Int J Obes (Lond) 2006;30:988-992.
22 Bedogni G, Malavolti M, Severi S, Poli M, Mussi C, Fantuzzi AL, et al. Accuracy of an eight-point tactile-electrode impedance method in the assessment of total body water. Eur J Clin Nutr 2002;56:1143-1148.
23 Tanner JM, Whitehouse RH. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Arch Dis Child 1976;51:170-179.
24 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-419.
25 Utriainen P, Voutilainen R, Jaaskelainen J. Continuum of phenotypes and sympathoadrenal function in premature adrenarche. Eur J Endocrinol 2009;160:657-665.
26 Schwandt P, Kelishadi R, Haas GM. First reference curves of waist circumference for German children in comparison to international values: the PEP family heart study. World J Pediatr

2008;4:259-266.
27 Jackson LV, Thalange NK, Cole TJ. Blood pressure centiles for Great Britain. Arch Dis Child 2007;92:298-303.
28 Ford AL, Hunt LP, Cooper A, Shield JP. What reduction in BMI SDS is required in obese adolescents to improve body composition and cardiometabolic health? Arch Dis Child 2010;95:256-261.
29 Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001;344:1343-1350.
30 Wunsch R, de Sousa G, Toschke AM, Reinehr T. Intima-media thickness in obese children before and after weight loss. Pediatrics 2006;118:2334-2340.
31 Schiel R, Beltschikow W, Radon S, Kramer G, Perenthaler T, Stein G. Increased carotid intima-media thickness and associations with cardiovascular risk factors in obese and overweight children and adolescents. Eur J Med Res 2007;12:503-508.
32 Pinhas-Hamiel O, Lerner-Geva L, Copperman NM, Jacobson MS. Lipid and insulin levels in obese children: changes with age and puberty. Obesity (Silver Spring) 2007;15:2825-2831.
33 Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Manuel Gómez J, et al. Bioelectrical impedance analysis-part II: utilization in clinical practice. Clin Nutr 2004;23:1430-1453.
34 Kalavainen M, Korppi M, Nuutinen O. Long-term efficacy of group-based treatment for childhood obesity compared with routinely given individual counselling. Int J Obes (London) 2011;35:530-533.
35 Schiel R, Beltschikow W, Radón S, Kramer G, Schmiedel R, Berndt RD, et al. Long-term treatment of obese children and adoloscents using a telemedicine support programme. J Telemed Telecare 2008;14:13-16.

Received May 24, 2011
Accepted after revision June 28, 2011